The Journal of HEREDITY

LILY CHROMOSOMES
(SEE PAGE 301)

Albino Lettuce

Short-Legged Turkeys

Developmental Defects in Twins

Medicine Men and the Pod Corn Gene

VOLUME 35 OCTOBER, 1944 NUMBER 10

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

DAVID FAIRCHILD, President Vice-President, SEWALL WRIGHT Secretary, C. E. LEIGHTY
Treasurer, J. H. KEMPTON

EDITORIAL BOARD

M. DEMEREC
H. J. MULLER

Managing Editor, ROBERT C. COOK

R. R. GRAVES

GEORGE M. DARROW
PAUL POPENOE

Members of the Council are ex-officio members of the Executive Board

COUNCIL

W. E. CASTLE, Emeritus Professor of Genetics, Harvard University, and Research Associate of the Carnegie Institution of Washington.
LEON J. COLE, Professor of Genetics, University of Wisconsin.
O. F. COOK, Bionomist (Retired), U. S. Department of Agriculture.
GEORGE M. DARROW, Horticulturist in Charge of Small-Fruit Breeding, Horticultural Field Station, Beltsville, Maryland.
DAVID FAIRCHILD, Agricultural Explorer (Retired), U. S. Department of Agriculture.
HARRY V. HARLAN, Senior Agronomist in Charge of Barley Investigation, U. S. Department of Agriculture.
J. H. KEMPTON, Agricultural Advisor to the State Department, Caracas, Venezuela.
W. V. LAMBERT, Associate-Director, Purdue University Agricultural Experiment Station, Lafayette, Indiana.
C. E. LEIGHTY, Agronomist in Charge of Dry Land Agriculture, U. S. Department of Agriculture.
HUGH C. MoPHEE, Chief of Animal Husbandry Division, U. S. Department of Agriculture, Washington, D. C.
MARCUS M. RHOADES, Department of Botany Columbia University, New York City.
SEWALL WRIGHT, Professor of Zoology, University of Chicago.

ADVISORY COMMITTEE

W. S. ANDERSON, Genetics Department, University of Kentucky, Lexington, Kentucky.
E. B. BABCOCK, Genetics Department, University of California, Berkeley, California.
E. A. BESSEY, Professor of Botany, Michigan State College, Lansing, Michigan.
A. F. BLAKESLEE, Professor of Botany, Smith College, Northampton, Mass.
W. E. BRYAN, Professor of Plant Breeding, University of Arizona, Tucson, Arizona.
RUSSELL W. DUCK, Editor, *The Rural New Yorker*. New York City.
R. A. EMERSON, Professor of Plant Breeding, Cornell University, Ithaca, New York.
C. P. GILLETTE, President, The Genetic Foundation, Fort Collins, Colorado.
C. M. GOETHE, Sacramento, California.
HARRISON B. HUNT, Professor of Zoology, Michigan State College, East Lansing.
H. S. JENNINGS, Emeritus Professor of Zoology, Johns Hopkins University, (Berkeley, Calif.).
D. F. JONES, Plant Breeder, Connecticut Agricultural Experiment Station, New Haven, Conn.
M. A. JULL, Professor of Poultry Husbandry, University of Maryland, College Park, Md.
F. B. LINFIELD, Director, Montana Experiment Station, Bozeman, Montana.
C. C. LITTLE, Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.
JAY L. LUSH, Professor in Animal Husbandry, Iowa State College, Ames, Iowa.
H. H. NEWMAN, Emeritus Professor of Zoology, University of Chicago, (Clearwater, Fla.).
T. S. PALMER, Expert in Game Conservation, U. S. Dept. of Agriculture, Washington, D. C.
JOHN H. PARKER, Director, Kansas Wheat Improvement Association, Manhattan, Kansas.
JOHN W. SCOTT, Professor of Zoology, University of Wyoming, Laramie, Wyoming.
A. D. SHAMEL, Physiologist, U. S. Department of Agriculture, Riverside, California.
GEORGE H. SHULL, Professor of Biology, Princeton University, Princeton, New Jersey.
EDMUND W. SINNOTT, Professor of Botany, Yale University, New Haven, Conn.
W. W. SMITH, Professor of Animal Husbandry, Purdue University, Lafayette, Indiana.
A. B. STOUT, Director of Laboratories, New York Botanical Gardens, New York City.
W. T. SWINGLE, Plant Physiologist (Retired), U. S. Department of Agriculture.
W. H. TOMHAVE, American Aberdeen-Angus Breeders' Association, Chicago, Ill.
H. E. WALTER, Professor of Biology, Brown University, Providence, Rhode Island.
H. J. WEBBER, Prof. Subtropical Hort., Citrus Experiment Station, Riverside, California.
E. N. WENTWORTH, Armours' Bureau of Agr'l Research and Economics, Chicago, Illinois.
P. W. WHITING, Zoological Laboratory, University of Pennsylvania, Philadelphia, Pa.

The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore, Md., and 1108 Sixteenth St., N. W., Washington, D. C. Address all communications regarding editorial matters to Room 205, 1108 Sixteenth St., N. W., Washington, D. C. Communications regarding the business management of THE JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to 1108 Sixteenth St., N. W., Washington 6, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 29, 1918.

Contents copyrighted 1944 by the American Genetic Association. Unauthorized reproduction of material from THE JOURNAL not permitted. The title "Journal of Heredity" is registered as a trade-mark in the U. S. Patent Office.

$3.50 a Year. 35c a Copy. Prices of back numbers on application.

The

Journal of Heredity

Vol. XXXV **OCTOBER, 1944** **No. 10**

CONTENTS

MEDICINE MEN AND THE PRESERVATION OF A RELICT GENE IN MAIZE
By Hugh C. Cutler 291

INHERITED SHORTENING OF THE LONG BONES IN THE TURKEY
By V. S. Asmundson 295

THE ORIGIN OF LILIUM TESTACEUM
By S. L. Emsweller and Maydelle B. Stewart 301

ONE-EGG TWINS WITH SPINA BIFIDA AND POLYDACTYLY
By H. H. Newman and W. B. Quisenberry 309

THE INHERITANCE OF CHLOROPHYLL DEFICIENCIES
By Thomas W. Whitaker 317

Notes and Reviews

FACTS ABOUT CANCER (Rev. of C. C. LITTLE: *Cancer, a Study for Laymen*) 294
"NO LAW OF HEREDITY"! 299
"NETWORK" VERSUS SEPARATE PEDIGREES 300
HUMANITARIAN EUGENICS 308
A STUDY OF RACE (Rev. of Contributions to the Genetics, etc., of *D. Pseudoobscura*) 314
INBRED SUNFLOWERS 320

OUR MEN NEED
★ BOOKS ★

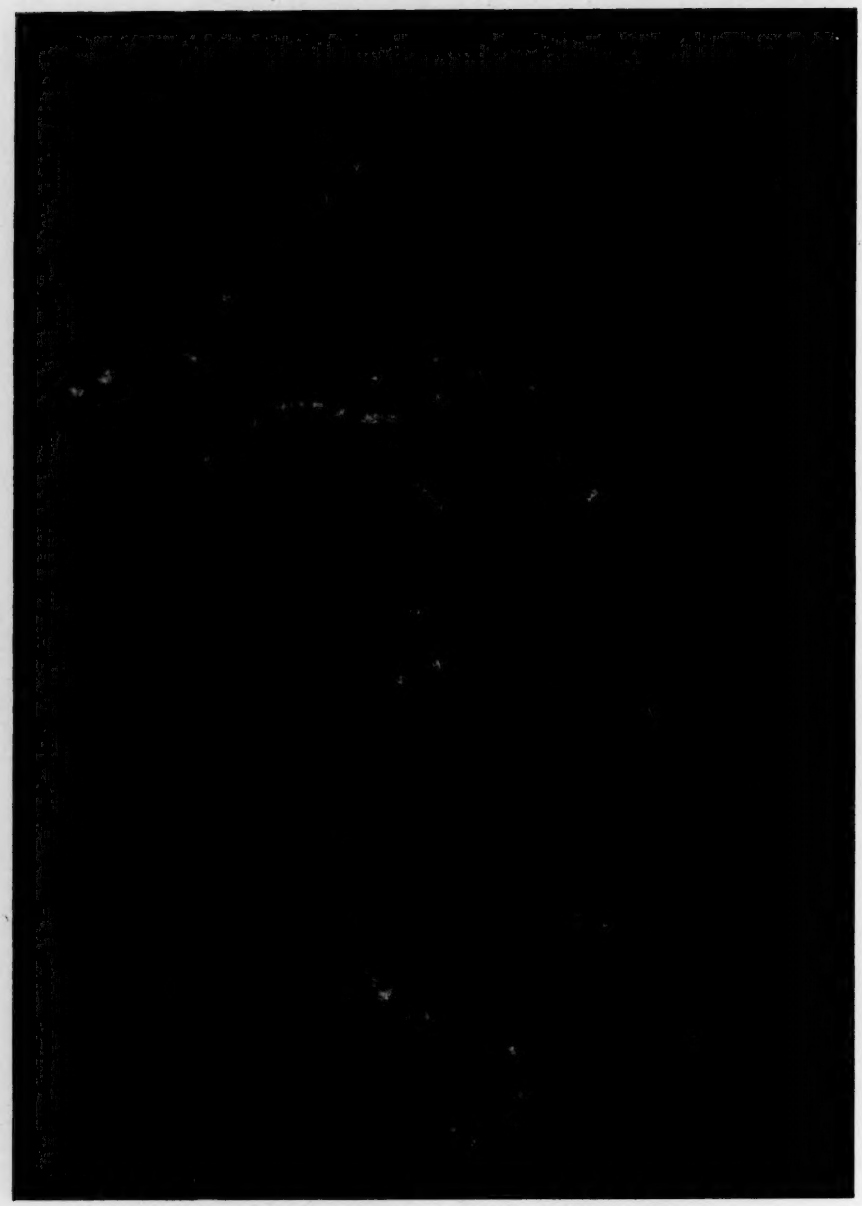
SEND
ALL YOU CAN SPARE

GIVE A BOOST
WITH A BOOK

Good books, in good condition, are wanted by the

1944 VICTORY
BOOK CAMPAIGN

for men in all branches of the service. Leave yours at the nearest collection center or public library.



KOCOPELLI, THE HUNCHBACKED FLUTE PLAYER

Figure 1

This petroglyph of the flute player is from the upper reaches of the Glen Canyon of the Colorado River in Utah. The same figure is frequently encountered in the pre-Columbian rock drawings of the southwestern United States. In this same region ancient ears of pod corn (Figure 4) have been found, thousands of miles from the assumed origin of this form in South America. This relic of the early evolution of maize is part of the stock in trade of the travelling medicine men of the Callalhuayo Indians of the Andes (Figure 2). Is it possible that Kocopelli of the Utah and Arizona canyons was a remote ancestor of the flute playing nomads of today who still carry an ear of pod corn in their packs?

MEDICINE MEN AND THE PRESERVATION OF A RELICT GENE IN MAIZE

HUGH C. CUTLER*

THE "HUNCHBACKED FLUTE PLAYER" OF TODAY

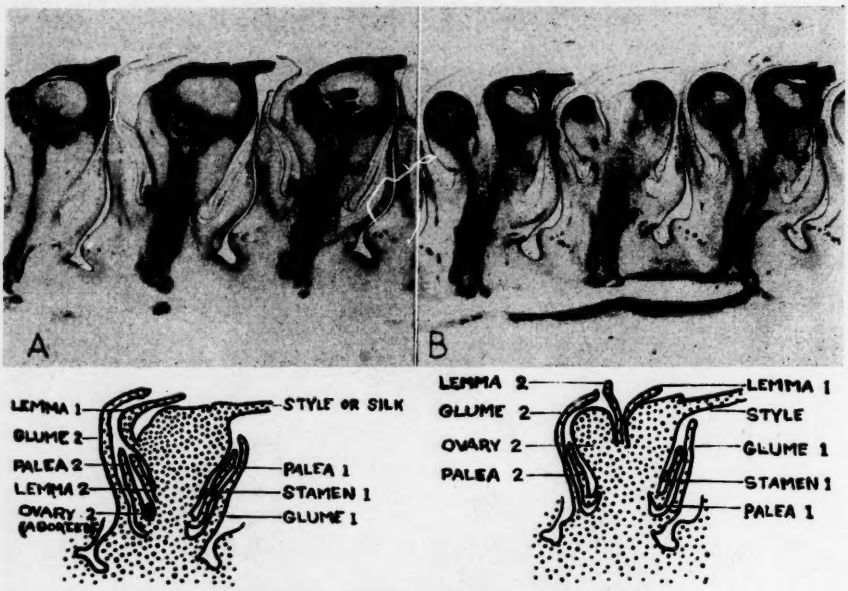
Figure 2

A Quichua Indian of Cochabamba, showing the carrying blanket and flute common throughout the Andean highlands. Possibly one of his ancestors, similarly equipped, carried pod corn to North America and became the legendary flute player, Kocopelli.

POD corn or tunicate maize is a dominant condition which has attracted the attention of botanists since it was first described. The covered grains have been considered a primitive character. Mangelsdorf and Reeves[3] and Brieger[1] have each reconstructed hypothetical wild forms of maize in which podded characters play a major role. An ear of pod corn is shown in Figure 4.

If the podded condition, which is dominant, really descended from original wild maize and has only changed in its aspects by the accumulation of modifiers, it is difficult to understand how it could have survived. Grains enclosed by the resistant enveloping glumes are not easy to clean and for all practical purposes would be undesirable in the Indian economy. Once selection against the podded condition

*Research Associate (on leave), Botanical Museum, Harvard University. This work is part of that done while a Fellow of the Guggenheim Foundation, 1942-43.



SINGLE AND DOUBLE MAIZE SPIKELETS

Figure 3

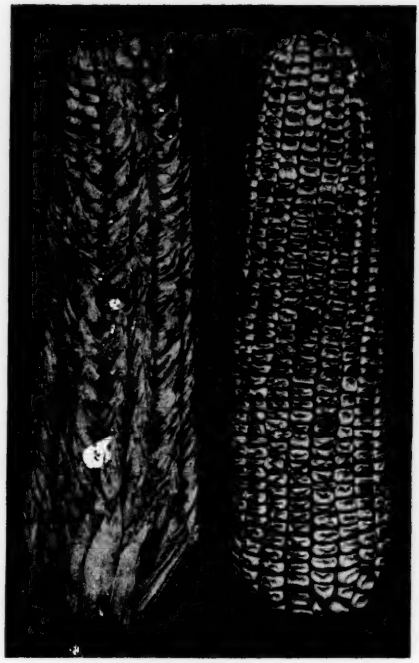
The microphotographs and diagrams show the difference between the usual formation of the maize spikelet (*A*) as compared with that of Country Gentleman sweet corn (*B*). Initially the maize spikelet has two flowers, the lower one remaining abortive, as is shown in the section of ordinary maize from Tarija, Bolivia (*A*). The grain arising when this second flower develops is pushed to one side, producing irregular rows. Photomicrographs made at the Genetic and Cytology Laboratory, Escola Superior de Agriculture Luis Queiroz, Piracicaba, Brazil. Magnification ×10.

was started, the character could be eliminated in a few years, for it is readily seen in the ear and the planters would probably learn to eradicate plants which had pod corn tassels. It is likely that this selection against pod corn was carried on, for in the past six years of work with Indian or part Indian agriculturists I have found that none of those questioned in this respect had ever encountered such an ear in his own fields.

Indian Plant Breeders

The American Indians, although far from expert plant breeders, were careful and consistent in their plantings. They selected only seeds of good quality from ears which met definite standards. There still exist traditions that the best ears must be placed before the gods after harvest time and taken out to be planted next season. Sometimes, as in Mexico, Guatemala, Bolivia, and the southwestern United States, the best ears are dressed in clothes and carried about in a ritual dance. Among the Indians of our Southwest it is occasionally said that seed is a sign to the gods so they will know what the planter wants.

Contamination of varieties is prevented by the difference in time of flowering of many varieties or by customs which demand that each kind of corn be planted at a different time of the year or in a separate plot. A dominant undesirable character easily visible in ear and tassel could be eliminated quickly in cultures where selection was practiced and one would expect to find it absent in modern plantings. Since



POD CORN
Figure 4

An ear of pod corn compared with an ear of ordinary corn. This character has no agronomic advantage. That it has not entirely vanished has been ascribed to repeated mutations. Perhaps the use of this odd form by medicine men has served to perpetuate and distribute it widely. (Photograph from James Kempton, this JOURNAL 1926.)

no wild maize which would provide a reservoir of the character has been found, it appeared that the present tunicate might be a recent mutation, but one which had occurred several times. It was therefore a surprise to find that, although tunicate maize was rarely, if ever, found in cultivated fields in Bolivia, practically all of the older Quichuas, especially the women, knew it by its name, *paca sara* (hidden maize). Dr. Martin Cárdenas[2] found that the grains are used as a remedy for *el aire* (the air), which may be tuberculosis, asthma, bronchitis or anything in which chest and breathing are affected. Pod corn could occasionally be found in the stocks of medicine men who travel about the country.

In the stock-in-trade of one of these wandering cure vendors in the Clissa (Bolivia) market, Dr. Cardenas and I found, besides an old and battered ear of pod corn with some grains missing, an ear of corn in which the lower as well as the normal upper flower of the spikelet had developed. This is the condition found in Country Gentlemen sweet corn of the United States (Figure 3). The additional grains are forced to one side or the other of the normal grain and the rows become characteristically irregular. While several different genes influence the development of the lower flower of the female spikelets, most of these genes are recessive as in the Bolivian material so far studied. These genes probably have many modifiers besides being affected by environmental conditions. Development of both spikelets is only selected against in varieties where straight rows are desired. As one would expect where irregular-rowed types of maize are common, the double grain spikelet is frequently found in Bolivia. The Quichuas call this type of maize *cuti sara* "turned maize," for the lower grain has the embryo on the side of the grain facing the butt while the upper grain has it on the side of the grain towards the tip of the ear). They ascribe to it the same curative properties as pod corn.

Most of these travelling medicine men are Callahuayo Indians who are taught from birth to know the virtues of many plants. Although their most common territory is the Andean highlands, they travel all over South America and even to Central America, carrying their packs of remedies. Their favorite musical instrument is the *quena*, a reed flute which often is found having a pre-Conquest arrangement of notes.

The flute, the pack of remedies slung over the back, and the long trips remind one of the rock pictures of the "hunchbacked flute player" occasionally called Kocopelli. This character apparently wandered from pueblo to

pueblo and did not confine his visits to any one group of our Southwestern Indians of about 1300 A.D. Along the tributaries of the San Juan River and in Glen Canyon of the Colorado River, his likeness in petroglyph and pictograph is especially common (Figure 1). Many of the flutes are pointed downward, eliminating the possibility of the instrument being a blow gun or a tube for symbolic cloud blowing or even a stick for swallowing. The hunched back is more deeply pecked in some of the petroglyphs and its position and shape suggest an Andean Indian carrying blanket (Figure 2).

In the Museum of Northern Arizona at Flagstaff is an ear of pod corn from Betatakin, a large pueblo ruin on a tributary of the San Juan River. To the writer it seems likely that the ancient Southwestern hunchback was actually a Callahuayo medicine man bringing to North America the character of tunicate maize which had not been introduced earlier in ears selected for food use. If this is the case, the beliefs of these Indians not only preserved this wild relict gene, but made possible for it to spread from its original home to all parts of the Americas.

Literature Cited

1. Brieger, F. G. *Revista de Agricultura*, (Piracicaba, Brasil) 18:409-418. 1943.

2. Cárdenas, Martin. Notas preliminares sobre la materia medica boliviana. Publicaciones de la Universidad de Cochabamba, Bolivia, 24 pp. 1943.

3. Mangelsdorf, P. C., and R. G. Reeves. *Texas Agric. Exp. Sta. Bull.* 574:1-315. 1939.

Facts About Cancer

CANCER is a disturbance of fundamental growth processes. Acquainting the layman with the facts about cancer should therefore give him enough biological background to see cancer in its total life context. A recent publication of the American Society for the Control of Cancer* does that job surpassingly. This is divided into three sections and fourteen chapters contributed by eleven outstanding leaders in biology and cancer research. The first section, authored by C. C. Little, and presenting the biological background, furnishes an insight into research methods, and discusses how cancer fits into the developmental pattern of the growing and aging organism. The Mendelian mechanism of heredity is clearly and simply explained in word and drawings, and the genetic component of cancer is discussed without alarmism. It sums up to a policy of cagy watchfulness. "This means that for the present at least and until further knowledge is obtained the individual whose parents died of cancer or in whose family the disease is frequent, should be alert, cautious and intelligent in detecting and reporting any sign or symptom of cancer in his own body. It does *not* mean that he should take a pessimistic or fatalistic attitude concerning his chances of having cancer. He is *not* doomed or condemned . . ."

The second section gives details on the diagnosis and treatment of cancer by specialists in cancer of the various organs of the body. The illustrations are helpful. As in fighting Japs, the reader comes to realize that eternal watchfulness may add years to one's life expectancy.

The reviewer recently saw a refractory sore on the neck of a neighbor. This arose from accidentally cutting a mole over two years ago. The wound had never quite healed. Two doctors had examined it and had tried unsuccessfully to get it to heal. Nobody had suggested that it could easily be an incipient cancer and that it should be laboratory checked with an eye to removal. Anyone who had read this book would not make this mistake. Even the layman himself would have been on his guard before a minor local growth upset had been translated by metastasis into cancer and another needless sacrifice to ignorance or indifference had resulted.—R. C.

"Cancer, a Study for Laymen," prepared for the Woman's Field Army of the American Society for the Control of Cancer, Inc. Pp. 122, illustrated with drawings and photographs. Price $1.00. Farrar and Rinehart, Inc., New York, 1944.

INHERITED SHORTENING OF THE LONG BONES IN THE TURKEY

V. S. ASMUNDSON

Division of Poultry Husbandry, University of California, Davis

INHERITED lethal or sub-lethal skeletal abnormalities have been reported in many species of domesticated animals and in man. The best known cases in cattle are the "bull dog" calves, which may be either dominant[6] or recessive.[13] In chickens several mutations are known which affect the length of the leg and wing bones. These include the extensively investigated creeper[12] and at least four others which affect the long bones.[2,7,9,10] While homozygous creepers usually die during the first few days of incubation, the other mutants ordinarily survive up to the time of hatching, and in the case of the chondrodystrophic mutation reported by Hays[7] in the Rhode Island Reds some of the embryos hatch but none survive more than a few days.

Pedigree breeding of turkeys was started much later than pedigree breeding of chickens. Moreover, pedigree breeding of turkeys is on a small scale compared to that of the domestic fowl. It is therefore to be expected that few lethals have not yet been found in turkeys. Nevertheless, two have been reported which are lethal or sublethal. These are the albino of Hutt and Mueller,[8] a sex-linked recessive sublethal, and short spine,[3] a skeletal abnormality which prevents hatching of the affected embryo.

Another sublethal mutation was obtained in 1937. A short-legged hen (Figure 5) was sent to me by Mr. Craig Wilson, Palmdale, California, who found it in a commercial flock of Bronze turkeys. An attempt was made to obtain others but none was found. For the sake of brevity these mutants, which are characterized by a shortening of the long bones of the legs and wings, will be referred to simply as "short."

When the short hen was mated to a normal male of known ancestry, all the progeny were apparently normal. F_2 progeny were raised the following year and in subsequent years. The data (Table I) show that the mutation is a simple autosomal recessive, but, as will be shown, the heterozygotes have shorter long bones than normal homozygotes. The deviation from the expected number (580.5 normal to 193.5 short) is 9.5 with a standard error of 12.0 — hence not significant. There were more known males than females, but here again the deviation from the expected sex ratio of 50.17 per cent males[1] of 2.11 ± 1.98 per cent is not significant.

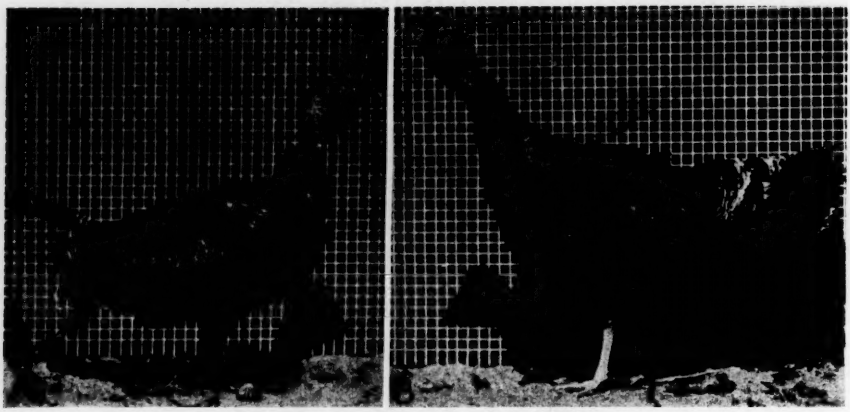
A Bronze male heterozygous for the mutant gene was mated to Black hens. A male out of this mating, heterozygous for the pattern genes (B = black; b = Bronze) and the genes affecting the long bones (for which the symbols S = normal and s = short may be used) was then mated to Bronze females of the constitution *bbSs*. The results (Table II) indicate that there is no linkage between the genes b and s since the percentage of short Bronze progeny was no greater than that of the short Black.

Viability of Short Turkeys

Linkage tests with the s gene are made difficult by the low viability of short poults. The hatchability of normal and short embryos during the five-year period for which records are available was as follows:

	Percent of embryos hatched	
Year	Normal	Short
1939	63.2	47.4
1940	75.0	26.3
1941	74.5	2.8
1942	80.8	18.8
1943	77.8	----
Average	74.3	19.1

Taking the combined data for all years, the percentage of short embryos



SHORT MOTHER, TALL DAUGHTER

Figure 5

The original "short" hen is shown on the left compared with her heterozygous daughter. The difference in stature is mainly due to difference in length of the shank (tarsometatarsus). The long bones of the wing are also affected, but the feathers conceal this difference.

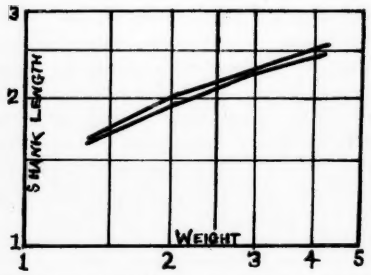
hatched was about one-fourth of the corresponding percentage for normal embryos. It should be kept in mind that the data are for classified embryos. All of the embryos dead before the tenth day of incubation and a few of the embryos that died before the twentieth day of incubation were not classified. The number that died from the tenth to twentieth day and were not classified was less than two per cent of all embryos alive at ten days of incubation; hence their omission has little effect on the tabulation. The viability of the short embryos up to the twentieth day of incubation was about the same as that of normal embryos or better than 90 per cent of embryos alive at ten days. Thus, like certain other mutations affecting the skeleton of birds, the short mutation does not reduce viability up to hatching time but does interfere with the emergence of the embryo from the shell.

The embryos that hatch are also handicapped. Of 25 short turkeys hatched only six have been raised. Undoubtedly more of them could be raised if kept by themselves where they would not have to compete with the longer legged and more agile normal poults. Actually, however, only about three per cent of the known short embryos were hatched and raised, which amply justifies calling the mutation a sublethal.

Changes in the Shape of Embryo Bones

Short turkeys are most readily distinguished from normal birds by the length of the tarsometatarsus (Figure 5). In Table II data are presented for the bones of embryos taken from the incubator on the 29th day. I am indebted to Mr. David Wittenberg, Jr., for the measurements and to Mr. Remo Navone for the analyses of the bones. The mutation did not have a consistent effect on the ash content of the bones; hence the percentages of ash in the bones are omitted. The embryos were paired by taking a normal embryo and pairing with a short-limbed embryo from the same hen. In each the embryos were fully formed and had the yolk sac completely inside the body cavity of the embryo. Normal embryos may have included both homozygotes and heterozygotes (*SS and Ss*), although the latter have slightly shorter and thicker shanks. The methods of measuring and analyzing the bones were the same as previously described.[2,3]

The data in Table III show that all



WEIGHT : SHANK : LENGTH RATIOS

Figure 6

Length of shank in centimeters plotted against body weight in grams for homozygous normal and heterozygous "short" turkeys at four-month intervals from four to 32 weeks of age. The congruence of the two lines demonstates that relative rates of growth are the same in these two groups. The difference in length of the leg bones was determined at an earlier stage of development.

the long bones of the leg are shorter in the mutants. Moreover, the femur, tibia, tarsometatarsus, and humerus were all significantly thickened ($P = <.001$). The radius and ulna were also thickened and at least in the case of the radius the difference from the normal may be regarded as probably statistically significant ($P = .015$ and $.05$). The total length of the neck vertebrae of the mutant was less and the vertebrae were wider than those of the normals, but these differences either were not significant or were of doubtful significance (length $P = .1$: width $P = .02$). The size and shape of other bones was not affected by the mutation.

With the exception of the femur, there was no significant change in the air dry weight of the bones. It is clear from the data for the weights of the bones that the increase in width or thickness of the affected bones more than compensated for their shortness. There is thus no dwarfing of the limb bones, merely a change in the length-thickness ratio. The changes are not the same for all the bones. In each case the first bone of the limb (femur or humerus) is affected less than the next more distal bone (tibia or radius and ulna). Thus the lengths of the bones from the mutants as a percentage of the length of the corresponding normal bones are as follows: femur — 85.6; tibia — 77.2; humerus — 89.2; radius — 85.4. Similarly calculated data for width were as follows: femur — 118.8; tibia — 121.4; humerus — 118.2; radius — 122.7. The tarsometatarsus was in this sample not more affected than the tibia. It is of interest to note that in the case of two mutations affecting the length of the limb bones in chicks[2,11] the tibia was shortened more than the femur, the radius (and ulna) more than the humerus.

Description of Mature Short Birds

Mature short hens have shanks a little over 12 cm. long or about 80 per cent of normal. The shape of the long bones of these hens is not noticeably abnormal, nor is there any apparent effect on the shape or relative size of the neck and head. Two of the three short hens were small. Three mature short males had shanks 14.9 cm. long, whereas those of heterozygous males are at least 2 and more typically 2.5 or 3 cm. longer. The shanks of homozygous normal males kept for breeding in 1943 varied from 17.2 to 23.2 cm., so that the length of the shanks of the heterozygotes was within the normal range (which is also true for the shanks of the females), but when paired with homozygous normal males of similar weight the shanks differed by about 1.6 cm. This is a decrease from the normal of about 7 per cent and a reduction in length of a little over 20 per cent in the case of mature short males. The short males had straight tarsometatarsi but the tibias were abnormally bent. The ash content was normal, 55 and 59 per cent of the air dry weight of the tibias. The males walked awkwardly and were unable to mate. The three males raised to maturity weighed 6600, 7800, and 8300 grams, or considerably less (by over 2000 grams) than normal males of the same strain. The abnormalities found in mature short turkeys, particularly the males, indicate that they are achondroplastic dwarfs.

Incomplete Dominance of the Normal (*S*) Gene

It is possible to segregate homozygous from heterozygous normals with some degree of accuracy when turkey poults are removed from the incubator. At 24 weeks of age the segregation can be relatively accurately made by measuring the shank (tarsometatarsus). An adjustment for differences in weight may be necessary in some cases since the length of the tarsometatarsus of turkeys is correlated with their weight.[4,5]

Data are available for length of tarsometatarsus and body weight of a few homozygous and heterozygous normal males and females at four-week intervals from 4 to 32 weeks of age. The data for females, plotted in Figure 6, show that there was no difference in the relative rate of growth of the tarsometatarsi from 4 to 16 weeks. The difference in the length of the tarsometatarsus was presumably established during the embryonic period.

Measurements on 12 mature normal hens which were tested by breeding and found to be heterozygous show that they differ significantly from homozygous normal hens in length of the tarsometatarsus but not in length of the keel of the sternum. The normal hens were, with one exception, from other strains which are known not to carry the mutation. The hens were paired on the basis of mature body weight, arrived at by averaging three weights taken at four-week intervals in January, February, and March. The bones were measured once. The averages and the values of *P*, the probability that the differences are due to chance, are shown below.*

The data show that the gene *S* for normal length of the long bones is incompletely dominant to *s*. The difference is so slight, however, that the heterozygotes would be classified as normal on the basis of appearance. Since birds with relatively short legs are favored by most turkey breeders, these heterozygotes would stand an excellent chance of being used in the breeding flock. The fact that this mutation has not been reported before and that additional cases have not come to notice indicates its rarity.

Summary

A short-legged Bronze hen, found in a commercial flock of turkeys, when mated to a normal male produced normal progeny. In the F_2 generation a good approximation to the ratio of three normal to one short was obtained with

Table I. F_2 progenies from matings of heterozygous parents.

	Normal				Short			
Year	Sex Males	Females	unknown	Total	Males	Females	unknown	Total
1939	27	39	2	68	10	9		19
1940	10	15	19	44	8	10	1	19
1941	60	65	114	239	34	36	1	71
1942	115	90		205	36	25		61
1943	23	11	2	36	11a	3		14
Totals	233	220	137	590	99	83	2	184

Table II. Progeny from parents heterozygous for short (Ss). The male was a black heterozygote (Bb). The female was bronze.

Plumage	Normal Obtained	Normal Expected	Short Obtained	Short Expected
Black	104	99	71	73
Bronze	99	99	30	33
Totals	203	198	61	66

Table III. Mean dimensions and weights of normal and short bones*

	Normal Length mm.	Normal Width mm.	Normal Air dry weight gm.	Short Length mm.	Short Width mm.	Short Air dry weight gm.
Femur	18.85	1.96	.0371	16.96	2.79	.0446
Tibia	26.62	1.87	.0557	20.54	2.57	.0594
Tarsometatarsus	19.76	2.01	.0422	16.88	2.76	.0440
Humerus	13.76	1.65	.0150	12.28	1.95	.0178
Radius	12.94	1.19	.0063	11.07	1.46	.0072
Ulna	12.44	0.90		10.95	0.96	
Scapula	12.62	1.74	.0061	12.07	1.72	.0065
Coracoid	7.86	1.49	.0063	8.50	1.52	.0070
Ilium	18.53	4.74	.0177	16.65	4.76	.0190
Ischium	9.08	1.45	.0062	9.47	1.70	.0088
Pubis	11.10	.41	.0020	11.82	.74	.0024
Neck vertebrae	43.30	3.77	.1850	37.08	4.39	.1908

*Based on 10 embryos except as follows: 7 embryos, 8 embryos, 9 embryos. Figures underlined for short-limbed embryos differ significantly from those for normal embryos (P = .01 or less).

*Bone Averages	Weight of bird gm.	Length of keel of the sternum cm.	(tarsometatarsus) Length of shank cm.
Homozygous normal	6264	15.54	15.35
Heterozygous normal	6263	15.67	14.14
Value of P	.9	.6	.001

about equal numbers of short males and females. The mutation is, therefore, a simple autosomal recessive.

No evidence was found of linkage between the gene *s* (short) and the gene *b* (Bronze plumage).

Hatchability and postnatal viability were so reduced by the mutation that only three per cent of all short embryos were hatched and raised; hence the mutation is sublethal.

Short embryos had significantly shorter long bones than normal embryos. The femur, tibia, tarsometatarsus, and humerus of the short embryos were thicker than the corresponding normal bones.

Heterozygous mature hens had slightly but significantly shorter tarsometatarsi than normal hens of similar weight.

Literature Cited

1. Asmundson, V. S. *Amer. Nat.* 75:389-393. 1941.
2. ———. *Jour. Hered.* 33:328-330. 1942.
3. ———. *Proc. Soc. Exp. Biol. Med.* 50:120-123. 1942.
4. ———. *Poult. Sci.* 23:21-29. 1944.
5. ———, and I. M. Lerner, *Poultry Sci.* 19:49-54. 1940.
6. Crew, F. A. E. *Proc. Roy. Soc. B.* 95:228-255. 1923.
7. Hays, F. A. *Amer. Nat.* 78:54-58. 1944.
8. Hutt, F. B., and C. D. Mueller. *Jour. Hered.* 33:69-77. 1942.
9. Lamoreux, W. E. *Jour. Hered.* 33:275-284. 1942.
10. Landauer, W. *Jour. Genetics* 31:237-242. 1935.
11. ———. *Storrs Agric. Exp. Stat. Bull. 233,* 45 pages. 1939.
12. ———, and L. C. Dunn. *Jour. Genetics* 2:3297-413. 1939.
13. Mohr, O. L. *Zeitschr. f. indukt. Abstamm. u. Vererb.* 41:59-109. 1930.

"No Law of Heredity"!

THE *Christian Science Monitor* occupies a notable position among American newspapers. Its coverage of world news is excellent and it is one in a very small minority of newspapers with a full-time science editor, who must sometimes be perplexed as to his status. For that reason its coverage of science news, and its discussion of scientific discoveries, compares very favorably with the run of science reporting in the daily press.

This well-deserved reputation is most unfortunate when it is traded on to attack well-grounded scientific principles. The leading editorial on page 6 of the August issue of the *Monitor*, headed "No Law of Heredity," denies that any principles of heredity are operative with regard to human beings. To quote briefly from this curious statement: "The supposition that man invariably inherits bodily disfigurements, as well as personally good or personally evil character traits from their ancestors is a fallacy. Man under God's control is formed and preserved by his Maker. In Mind's uninterrupted and unobstructed continuity of unfoldment, all of God's ideas are conceived in beauty, grandeur, order, and perfection. When parents cease to ascribe inherited characteristics to their children, to bind them with having inherited the good or the bad character traits of either side of their families, children will be healthier and happier. . . . Spiritual man has never become material. When one has succeeded in conquering the belief in heredity in one's own thought, it is possible to prove that no deterrent such as family anxiety, friendly concern, or medical diagnosis can interfere with one's inherent right to spiritual progress, growth, and dominion." An instance is cited of curing a young man of over-obsession with his ancestors,—a thoroughly commendable undertaking. A second case of multiple escapes from hereditary defects is more interesting. A young woman was reported cured of what was said to be an incurable hereditary nervous illness through one Christian science treatment. "This healing took place 23 years ago and was permanent. In later years she was able to overcome poor eyesight, and was also saved from the inherited tendency of poor hearing."

It is precisely in the field of ill-defined, but none the less very real "nervous" and psychosomatic diseases that "faith healing" sometimes succeeds. To claim that one can be saved from "an inherited tendency of poor hearing" which has not become manifest only demonstrates a complete ignorance of genetics. How do we know the affliction was actually there in the first place?

One suffers from an embarrassment of riches in replying in detail. The *Monitor's* position reaches its ultimate absurdity in the case of twins, one of whom is albino and the other normally pigmented. Recent discoveries regarding the *Rh* factor might be cited as constituting controlled experiments of the view propounded in this article. The hereditary nature of infant jaundice and other manifestations of Rh incompatibility have only very recently been recognized. Physicians have in more than one instance insisted that couples who have had one affected child run no risk in having additional children; but this did not prevent disaster. In such a situation nobody "believed" in the operation of a "law of heredity," because it was not supposed to operate. Yet the outcome was just the same as though all the facts were known.

While such attacks merely amuse the well informed they may do considerable harm through misleading those who have a right to be warned against the transmission of hereditary defect. When spurious arguments lead to the perpetuation of monstrous or dehumanizing abnormalities those who give currency to unfounded beliefs take a great responsibility. In dealing with the problem of pathological heredity, aggressive ignorance is no defense, and those who do mislead the uninformed should be held accountable. R. C.

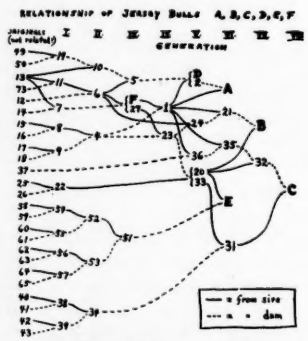
A "Network" Versus Separate Pedigrees

To the Editor:

The six pedigrees of Jersey cattle on Page 197 of the July issue have proved to be so intriguing to me and associates that I actually decided to see what the triangles, squares, and eggs signified. Translating the data from the Oriental (right-left) order to Occidental (reading from left to right), and avoiding senseless repetition, I arrive at the enclosed network of descent. The method is scarcely original with me, since G. H. Shull (*Genetics* 11.204, 1926), and Sewall Wright (*Jour. Agric. Research* 26: 165, 1923), have employed very similar principles. Which would you prefer—six pedigrees or one network?

W. F. Hollander

Carnegie Institution,
Cold Spring Harbor, L. I., N. Y.



SIX-PEDIGREE NETWORK

The editor votes for networks and commends same to expectant authors gestating still embryonic or preembryonic charts. It might be pointed out that the network technique violates most of the "unwritten conventions" of livestock pedigree compilation as described by Dr. Haines in this Journal last year (Vol. 34, p. 152). The "network" not only reads in the opposite direction but the relative locations of sires and dams become a matter of convenience, not of convention. Is it possible thot the rather slow penetration of genetic concepts into the thinking of some breeders has been partly due to a failure to use some such synthesizing system of "networks"?

THE ORIGIN OF LILIUM TESTACEUM

S. L. Emsweller and Maydelle B. Stewart*

THE Nankeen lily (× *Lilium testaceum*† Lindl.) is reported to have been found in 1836 in a bed of mixed *L. martagon* L. growing in a German nursery.[4] For a time it was thought to be a new species from Japan, but lily specialists soon considered it a hybrid between *L. candidum* L. and *L. chalcedonicum* L. It was not, however, until much later that any attempt was made to make the cross and verify the alleged parentage. According to a note by Elwes,[3] Tevor Clarke, in 1880, produced a lily hardly distinguishable from × *L. testaceum*, by fertilizing *L. chalcedonicum* with the pollen of *L. candidum*. Two years later, according to Weston,[15] H. P. Blackmore made the same cross and produced several seedlings which varied in both flower color and shape. One closely approached *testaceum*, differing only in having petals that were not quite so long or twisted. Recently F. J. Jones[5] reported on the results of crossing *L. chalcedonicum* var. *maculatum* Stoker with *L. candidum*. One seedling was flowered and, while closely resembling *testaceum*, it differed in being lighter in color, almost white outside, and without the slight red tinge. Later Jones sent small seedlings of the same cross to Major F. C. Stern[10] who grew them to flowering and described them as follows: "Most of the seedlings are an orange-apricot color and one is almost the color of × *L. testaceum*. All the flowers are the shape of the flower of *L. chalcedonicum*, about 2¾ inches across, with each petal if pulled out about 2¾ inches long."

As pointed out by Grove,[4] there may be more than one clon of *testaceum*. This is based on the reports of some *testaceum* plants bearing flowers with absence of markings on the petals. All the synthetic *testaceum* forms so far reported differ in some way from one another as well as from the type accepted for the hybrid. The stocks of this lily now available are all believed to be infected with virus. While insofar as is known the virus does not affect the vigor of this lily, aphids readily transmit the virus to less tolerant species, thus making *testaceum* one of the lilies to avoid if many other species are to be grown nearby. Therefore it is important to ascertain the exact parents so that new virus-free stocks may be made available. In the present paper cytological data are presented which show the existence of several types of both *candidum* and *chalcedonicum*, and that *testaceum* as constituted probably arose from a cross between certain forms of these two species.

Materials and Methods

Lilium chalcedonicum belongs to the Martagon section of the subgenus *Eulirion*. It is, according to Stoker[14] indigenous to Greece, being reported from Thessaly, Mt. Olympus, Euboea, and the Peloponnesus. There is a recognized form of the type in which the raised spots within the throat are colored purple. This form has been given the varietal name *maculatum*. The five *chalcedonicum* bulbs examined by us were furnished by George Slate from a group of seedlings. These five did not vary in any noticeable way from the description of the type.

Lilium candidum is one of the oldest members of the genus. It belongs to the Leucolirion section of the subgenus *Eulirion*. At the present time it is being grown in almost all parts of the world. There has been considerable speculation

*Principal horticulturist and formerly assistant scientific aid, respectively, Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, United States Department of Agriculture, Beltsville, Maryland.

†The sign of multiplication is inserted before the species name to indicate a hybrid species.

as to its native habitat and the problem is still unsolved. According to the Abbé Souillet[9] it probably originated in Asia Minor. This assumption was based on information from correspondents that a *L. candidum* grew wild there in great numbers. It was described as a short lily with small closed flowers, apparently highly fertile. This, however, may be the well-known fertile variety Salonikae which was found growing in the hills near Salonika, Greece. The Abbé Souillet describes seven other forms of *candidum* in addition to those already mentioned. These are: (a) the ordinary garden form of Europe which grows taller than Salonikae, is self sterile, and bears large, well-opened flowers; (b) the variety Charles X which reaches a height of two meters, bears very large, pure-white, flowers and is self sterile; (c) variety *peregrinum,* with narrow petals, separated at the base, and a dark, almost black stem; (d) variety *spicatum,* which bears very abnormal flowers, commonly considered a double form; (e) variety *foliis variegatis,* with variegated foliage; (f) variety *peregrinum fasciatum* which is reported to produce annually a broad fasciated stem; and (g) variety *purpureo-striatum,* in which the flowers, stems, leaves, and bulb scales are striped with purplish red. In addition to these forms Stoker[13] described a *candidum* that had been sent to England from Afghanistan. From his description it is possibly a new variety of *L. candidum* and may be closely related to the original wild form. Not enough comparative data are available to determine whether or not it actually differs from one of the nine forms listed by the Abbé Souillet.

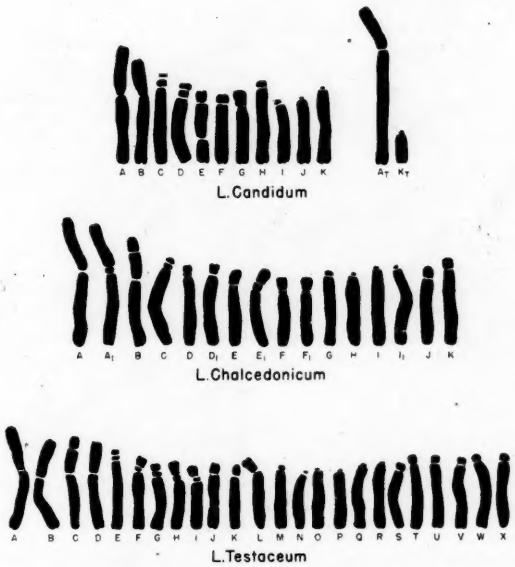
The *Lilium candidum* bulbs used in this investigation were secured from several sources. The three Salonikae bulbs were from George Slate, who also sent us eight seedlings from a cross between the ordinary form of *candidum* found in gardens at Geneva, New York, with the Salonikae form. His seedling No. 84 was selected from a population produced by crossing two *candidum* clons which originated from interpollinations among a group of *candidum plants.* We are also indebted to him for two bulbs of *L. candidum* var. *purpureo-striatum* which he secured from J. Averall, Darwin Nursery, Sulphur Creek, Tasmania. Twelve bulbs were secured from Vaughan's and the four bulbs examined appeared to be members of a clon. Two bulbs were sent by F. P. McWhorter who secured them from stocks grown in the vicinity of Corvallis, Oregon. They are listed as Oregon 1 and 2.

Six *testaceum* bulbs were secured from W. N. Craig of Weymouth, Massachusetts. They produced plants that appeared to be identical and no doubt were members of a clon.

The morphology of the chromosomes was determined from root tip smears. The excised root tips were first dropped into a 0.2 per cent aqueous solution of colchicine for 30 minutes. This technique, described by Burrell,[2] is a modification of that of O'Mara,[8] in which attached root tips were treated. It was also used effectively on root tips of *L. tigrinum* Ker-Gawl. by Stewart and Bamford.[12] The best results were secured with our material when the excised root tips were washed in water for from 15 to 20 minutes following treatment in colchicine. They were fixed in 3:1 acetic acid and absolute alcohol solution for 20 to 24 hours, then stored in 85 per cent alcohol until used.

Results

The *Lilium chalcedonicum* chromosomes are shown in the idiogram of Figure 7. Each of the five plants had 24 chromosomes, but there were numerous cases of heteromorphic pairs so that 16 different chromosomes were distinguished. In Table I is listed the frequency of each type of chromosome for each of the five karyotypes. All the chromosomes were not found in any one plant, nor did any two plants have identical chromosomes sets. As is shown in the table there were a few cases in which there was some question concerning the presence of a secondary constric-



THE CHROMOSOMES OF THREE LILY "SPECIES"

Figure 7

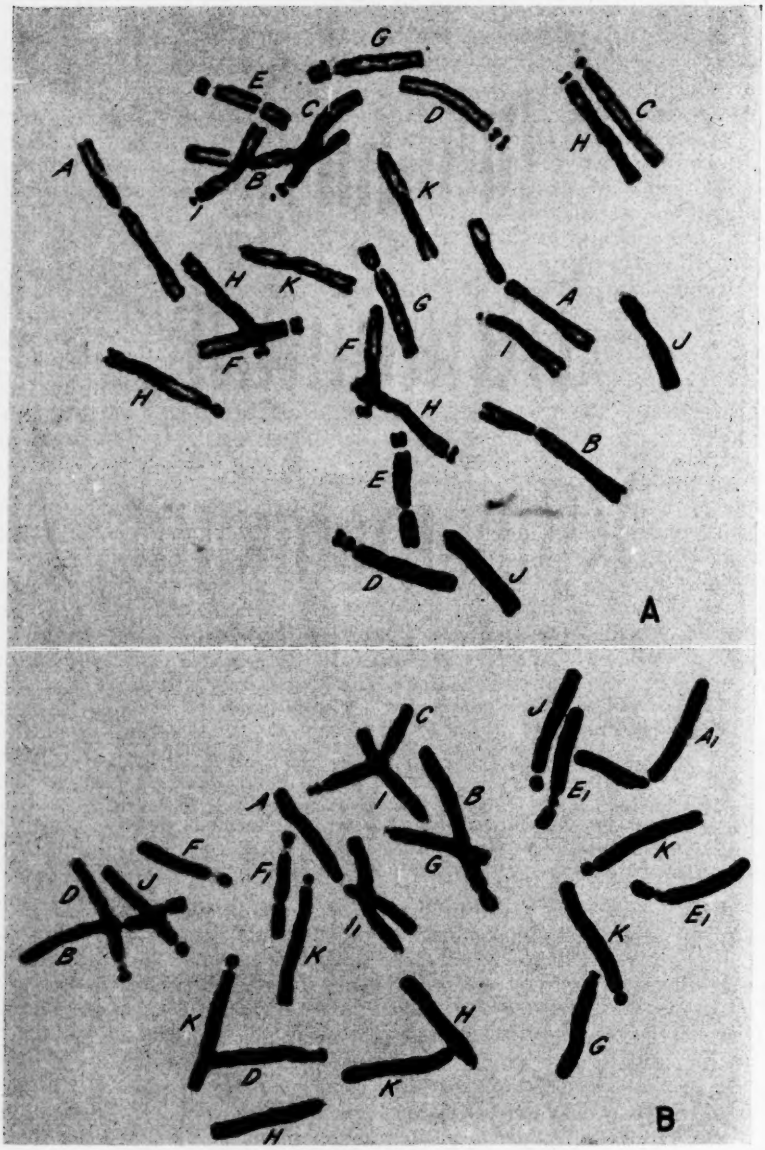
This chart shows idiograms of the chromosomes of the two species *Lilium candidum*, and *L. chalcedonicum*, and of the hybrid species "× *L. testaceum*." Each of the five plants of *L. chalcedonicum* studied had 24 chromosomes, but the 16 types here shown were distinguished. Not all of these were found in any one plant. The *testaceum* chromosomes designated *T, U, V, W, X* probably originated from *candidum H*-chromosomes, and *chalcedonicum K*-chromosomes. They cannot be distinguished morphologically.

tion. It is possible that a constriction was actually present and simply obscured in some instances. The photomicrograph in Figure 8*B* is from *chalcedonicum* plant No. 3. It includes 14 of the 16 types of chromosomes, lacking only D_1 and *E*.

In the idiograms of Figure 7 the primary constriction is the lower one except in the case of *Lilium chalcedonicum* F_1 and I_1, *L. candidum E*, and *L. testaceum I*. The secondary constriction in I_1 differs from the other secondary constrictions in that a nucleolus is not formed at that point and the constriction is never attenuated. In fact it is plainly visible in only a small per cent of the cells. It is clearly seen in Figure 8*B*. Similar constrictions are frequently observed in the other chromosomes but have not been demonstrated to be regular features of the chromosome morphology.

In making comparisons in length between chromosomes only those that are completely free of turns and coiling of the two chromatids can be used. Such comparisons in the five *chalcedonicum* plants show that in spite of the high frequency of heteromorphic pairs the differences between pairs are only in the presence or absence of secondary constrictions.

The high frequency of heteromorphic chromosome pairs encountered in *chal-*



CHROMOSOMES OF PUTATIVE PARENT SPECIES

Figure 8

Photomicrographs of mitotic metaphase chromosomes in root tips of: (*A*) *Lilium candidum* (Slate seedling No. 4); (*B*) *L. chalcedonicum* (Plant No. 3). These plants have a chromosome content which could combine to give a *testaceum*-type hybrid.

cedonicum is certainly unusual. The plants were from a seedling population grown at Geneva, New York, by George Slate. It is probable that the seed came from several *chalcedonicum* plants, but there is no way of checking this since the seed was purchased from a dealer several years ago. As mentioned earlier, the plants were all typical of *chalcedonicum*. Unfortunately they were all lost when they were attacked by basal rot after flowering.

In all the *Lilium candidum* forms except var. *purpureo-striatum* there were 11 readily distinguishable chromosome types. They are shown in the idiogram of Figure 7 and the photomicrograph of Figure 8*A*. The variety *purpureo-striatum* evidently originated following a simple translocation involving chromosomes *A* and *K* with *A* acquiring slightly over half the long arm of *K*. Since the centromeres were not affected, the modified A_t and K_t chromosomes behave normally in mitosis and have been perpetuated in this clon. The two unmodified *A* and *K* chromosomes are shown in the *candidum* cell in the photomicrograph Figure 8*A*, and chromosomes A_t and K_t resulting from the translocation are shown in Figure 10*B*. The A_t chromosome is one of the longest yet reported in plants.

In Table I the 16 plants of *L. candidum* observed by us are grouped into four types according to the morphology of their chromosomes.

In Type I, which includes three Salonikae plants and four Slate seedlings, the genom is balanced, with two each of ten of the chromosome types and four of the *H* type. This does not mean that these plants are tetrasomic for *H*, since identical morphology does not necessarily denote genic homology. As mentioned earlier, the four seedlings from Slate are from a cross between Salonikae and a Geneva form of *candidum*.

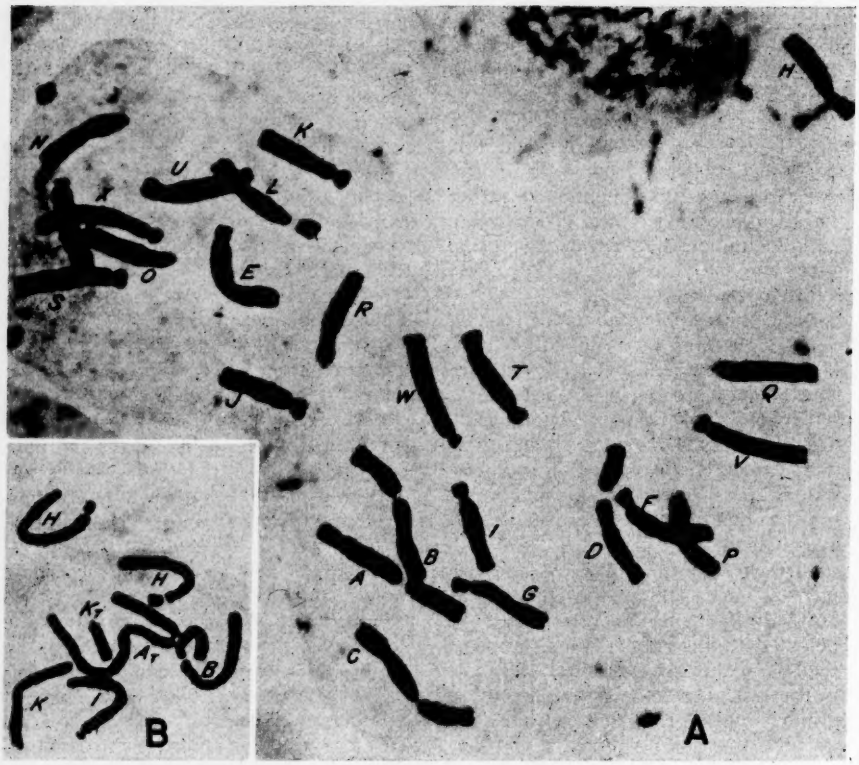
In Type II are included three other Slate seedlings. These differ from Type I in that one of the *C* chromosomes has lost its secondary constriction and is classed as an *H* chromosome. The presence of this constriction is, in fact, the only morphological feature which distinguishes the *C* and *H* chromosomes. In the photomicrograph of Figure 8*A*, this difference is clearly seen in the upper right-hand corner.

In Type III there are two other Slate seedlings, four bulbs from Vaughan's (all identical), and the two bulbs from Oregon. In all of these bulbs one of the *C* chromosomes lacked the secondary constriction. The plants also had three *J* chromosomes and only one *I*. The only observable difference between these latter two chromosomes is in the size of the short proximal arm. This may be clearly seen in the photomicrograph of Figure 8*A*. Of all the heteromorphic pairs in both parental types, this is the only one that involves an alteration in the measurable amount of chromatin material present in the genom.

Type IV includes only the translocation form, var. *purpureo-striatum*.

Very little has been published on the details of chromosome morphology in *Lilium candidum* and nothing on *L. chalcedonicum* or *L. testaceum*. Nemec,[7] working with a form of *candidum* secured from Italy, reported a heteromorphic pair of chromosomes, but did not attempt to distinguish the other chromosomes from one another. In the heteromorphic pair one chromosome had an appendage at its distal end. In his figure 8 this appendage is attached by a thread to the main body of the chromosome. It is rather large but it is not possible to determine its length in relation to the remainder of the chromosome.

Since all lilies may be propagated by asexual means and are usually so handled, the perpetuation of clons with heteromorphic chromosomes such as were found in *chalcedonicum* and *candidum* is easily understood. In this genus there is also a high frequency of species with extra chromosomes or fragments. Stewart[11] examined 86 species of *Lilium* and found six with one or more extra chromosomes or fragments. In all cases the morphology of the extra chromosomes differed from all of the



CHROMOSOMES OF TESTACEUM AND OF A TRANSLOCATED CANDIDUM

Figure 9

A shows a photomicrograph of a mitotic metaphase plate in a root tip of the assumedly hybrid species × *Lilium testaceum*. The chromosomes are labelled as in figure 8. *B* shows a section of a mitotic metaphase in a root tip of the variety *purpureo-striatum* of *L. candidum*. Here can be seen the A_t and K_t chromosomes. These were derived by a translocation from the *K* chromosome to the *A* chromosome. Note also the unmodified *K* chromosome.

normal 24 chromosomes. In at least one of these species, *L. pumilum* de Cand., some of the plants were known to be seedlings so that the fragments and extra chromosomes were transmitted in the gametes. Beal[1] also reported gamete transmission of a fragment in *L. willmottiae* Wils., and we have found a fragment in one of our seedlings of *L. henryi* Baker. Possibly a more critical examination of chromosome morphology in other lily species may show a condition similar to that of *chalcedonicum* and *testacum*.

The idiogram for *Lilium testaceum* is shown in Figure 7. The *testaceum* chromosomes *T*, *U*, *V*, *W*, and *X*, which had their probable origin from *candidum* *H* chromosomes and *chalcedonicum* *K* chromosomes (Table II), are morphologically indistinguishable. For this reason they afford a convenient standard of reference for comparing lengths of the other chromosomes of *testaceum* with the chromosomes of *candidum* and *chalcedonicum*. All the chromosomes in *testaceum* which have been selected as having their probable origin in *candidum* show the same length ratio to chromosomes *T*, *U*, *V*, *W*, and *X* that the cor-

responding chromosomes in *candidum* do to chromosome *H*. Likewise, all which have been selected as having their probable origin in *chalcedonicum* show the same length ratio to chromosomes *T, U, V, W,* and *X* that the corresponding chromosomes in *chalcedonicum* do to chromosome *K*. The only chromosome differences from the parental types that can be distinguished morphologically are the differences in the position of the secondary constrictions in the short arms of chromosomes *F* and *I* of *testaceum* as compared with chromosomes *D* and *F* of *chalcedonicum*. Taking into consideration the wide variation in position of secondary constrictions in the five plants of *chalcedonicum* that we were able to obtain, these differences do not seem serious enough to invalidate the theory that the chromosomes originated in *chalcedonicum*.

A photomicrograph of the chromosomes of *testaceum* in a root tip cell is shown in Figure 9*A*. The probable origin of each chromosome is given in Table II. It has been concluded that a *candidum* of Type III and a *chalcedonicum* similar to plant No. 3 were probably involved in the original cross which produced *testaceum*. This is suggested by the absence of a chromosome in *testaceum* corresponding to the *candidum C* chromosome and because of the presence

-12-

Table II. Probable origin of Lilium testaceum chromosomes

Testaceum chromosomes	Chromosomes from candidum	Chromosomes from chalcedonicum
A		A
B	A	
C		B
D	B	
E		C
F		D (modified)
G	D	
H		E_1
I		F (modified)
J	E	
K	F	
L	G	
M		G
N		
O	J (2)	H (1)
P		
Q	K	I
R		
S		J
T		
U		
V	H (3)	K (2)
W		
X		

Table I. Frequency of chromosome types in Lilium chalcedonicum and L. candidum

Plant No.	A	A_1	B	C	D	D_1	E	E_1	F	F_1	G	H	I	I_1	J	K
								Chalcedonicum								
1	1	1	2	-	1	1	1	1	1 or 2	1 or -	2	2	1 or 2	1 or -	2	6
2	-	2	2	1	2	-	2	-	1	1	2	2	2	-	2	5
3	1	1	2	1	2	-	-	2	1	1	2	2	1	1	2	5
4	-	2	2	1	1	-	1 or -	1 or 2	2	-	2	2	1 or 2	1 or -	2	5
5	-	2	2	1	2	-	-	2	2	-	2	2	2	-	2	5

		A	B	C	D	E	F	G	H	I	J	K	A_t	K_t
		Candidum												
Salonikae 1, 2, 3; Slate sdlgs. 1, 2, 4, 6	Type I	2	2	2	2	2	2	2	4	2	2	2		
Slate sdlgs. 3, 7, 84	Type II	2	2	1	2	2	2	2	5	2	2	2		
Slate sdlgs. 5, 8; Vaughan's Oregon 1; " 2	Type III	2	2	1	2	2	2	2	5	1	3	2		
Purpureo-striatum	Type IV	1	2	1	2	2	2	2	5	1	3	1	1	1

of only one chromosome like *M* in *testaceum*, which probably came from *chalcedonicum G* rather than from *candidum I*, since none of the *chalcedonicum* plants examined lacked a *G* and had an extra *H* chromosome. The presence of the *chalcedonicum A* and *C* chromosomes in *testaceum* also confirms this assumption. It is of course quite possible that a *candidum C* chromosome entered into the cross and the secondary constriction does not show in the hybrid due to the phenomenon of amphiplasty.[6] It is also possible that further examination of other *chalcedonicum* individuals would reveal some with a heteromorphic *G-H* pair similar to *candidum I-J* from which the hybrid could have received either *G* chromosomes or an extra *H* type. Since the *candidum* with two heteromorphic pairs is so common, the latter supposition is hardly necessary.

Summary

The morphology of the chromosomes of five *Lilium chalcedonicum*, sixteen *L. candidum*, and six *L. testaceum* plants was determined from root-tip smears. A high frequency of heteromorphic chromosome pairs was found in the *cholcedonicum* plants. The *candidum* plants also had some heteromorphic pairs. It was possible to assort the *candidum* plants into four groups, each varying from the other three in the number of certain chromosomes present. One type, *L. candidum* var. *purporeo-striatum*, had originated following a translocation of the major portion of one arm of the *K* chromosome to the long arm of the *A* chromosome.

The chromosomes of *Lilium testaceum* indicate that it probably arose from a cross between a *chalcedonicum* plant having a chromosome constitution similar to the plant designated as No. 3 in this paper and a *candidum* plant of Type III.

Literature Cited

1. Beal, J. M. *Botanical Gazette* 103:617-619. 1942.
2. Burrell. P. C. *Stain Technology* 14: 147-149. 1939.
3. Elwes, John H. A Monograph of the Genus Lilium. Taylor and Francas, Fleet St., London. 1880.
4. Grove, A. *Gardeners' Chronicle*. Sept. 17, p. 212, 1932.
5. Jones, F. J. *Royal Horticultural Society Lily Year-Book*, p. 115-116. 1932.
6. Navashin, Michael. *Cytologia* 5:169-203. 1934.
7. Nemec, B. Z. *Věstniku Král Ces. Spol. Nauk. Tr.* II. Roc. 1935.
8. O'Mara, J. G. *Journal of Heredity* 30: 35-37. 1939.
9. Souillet, Abbé. *Gardeners' Chronicle* 95:230-231. 1934.
10. Stern, F. C. *Royal Horticultural Society Lily Year-Book*, p. 124-125. 1936.
11. Stewart, R. N. *Botanical Gazette* 104:620-626. 1943.
12. ————, and Ronald Bamford. *American Journal of Botany* 30:1-7. 1943.
13. Stoker, Fred. *Royal Horticultural Society Lily Year-book*, p. 124-125. 1936.
14. ————. *Royal Horticultural Society Lily Year-book*, p. 86-107. 1938.
15. Weston, T. A. *Gardeners' Chronicle*. Oct. 22, p. 300. 1932.

◆

Humanitarian Eugenics

Eugenic policies sometimes have apparently irreconcilable purposes: to raise the general level of hereditary endowmnt, but without disregard to the pressing needs of mankind today. Often there is nothing to be done but to follow the dictates of our common humanity and as far as possible to palliate the dysgenic consequences by negative eugenic measures. Sometimes there is no real conflict and we can meet the claims of the future while taking good care of the present. It is true that some humanitarian measures are potentially dysgenic, but every well-conceived eugenic measure, without exception, is also humanitarian in the narrower sense of the term. The point set forth in the *Eugenics Society's* statement of aims and objects could be supported in detail without any reference to their long-term effects. These policies need no other justification than the contribution they offer to health and happiness here and now.—*Eugenics Review* (London), April 1944.

ONE-EGG TWINS WITH SPINA BIFIDA AND POLYDACTYLY

H. H. NEWMAN AND W. B. QUISENBERRY*

ON April 13, 1943, female twins were born to a colored woman 24 years old in the neighborhood of Crewe, Virginia. The birth was spontaneous under the supervision of an experienced and intelligent midwife, who, after a careful examination of the afterbirth, destroyed it. She reported to Dr. Quisenberry that the placenta was unquestionably single (not in any way constricted or divided), with two distinct umbilical cords. The two babies had been enclosed within a single chorion and amnion with nothing in the way of a partition separating them. On this basis alone, if the report of the midwife be regarded as accurate, the babies must be diagnosed as one-egg twins.

In addition to this, certain striking concordances with respect to rare and peculiar conditions in the twins would warrant a monozygotic diagnosis were nothing known about the fetal membranes. The twins were alike in the following respects:

a. They were both female.
b. Each weighed exactly four pounds at birth.
c. Each had two erupted upper incisor teeth at birth.
d. Each was afflicted with an extensive spina bifida involving the whole lumbar region and with myelomeningoceles.
e. The meningeal sacs leaked cerebrospinal fluid before death.
f. The lower extremities of each exhibited spastic paralysis.
g. Each passed urine and feces shortly after birth.
h. They were essentially identical in form and features except for discordance in polydactyly.

The only difference between the twins that was noted by the physicians in charge was that twin *A* had six digits on the right hand while twin *B* was normal in both hands and feet. The extra digit on *A*'s right hand was a normal thumb entirely separate from the somewhat larger, inner thumb, and had the usual number of joints and a well-developed thumb nail.

The twins were born a little later than the expected date and were in an advanced stage of development as evidenced by the eruption at birth of two teeth. This is in contrast with the fact that a large majority of twins are born prematurely.

Fortunately, considering the seriousness of their defects, the babies did not live long. Twin *A*, the polydactylous one, would take neither breast nor formula and died after thirty-six hours; while twin *B*, although she took the formula freely, expired sixty hours after birth.

The above data are all that we have that seem pertinent to the present discussion. Dr. J. A. B. Lowry also made a careful examination of the twins and confirms all the data reported by Dr. Quisenberry. It is regrettable that no photographs were made either of the

*On May 9, 1943, I received a letter from Dr. Walter B. Quisenberry, Assistant Surgeon, U. S. Public Health Service, located at the Southside Health District, Farmville, Virginia. Dr. Quisenberry was familiar with my studies of twins and thought I might be interested in an unusual pair of twins that had come under his observation. He therefore gave me a brief account of the case and asked whether I knew of any similar case having been reported in medical literature. He himself, he said, was writing up the case for a medical journal. I replied that, so far as I knew, the case was unique, was highly significant in several respects from a genetical standpoint, and that it deserved to be discussed and published in *The Journal of Heredity*. Dr. Quisenberry welcomed this suggestion and commissioned me to prepare a paper on the case and send it in to the Journal. He also sent me a full report on the twins and subsequently, at my request, investigated their family pedigree. The publication of this paper has been approved by the Surgeon General of the U. S. Public Health Service.—H. H. N.

placenta or of the twins, but the facts seem sufficiently well authenticated to warrant a theoretical discussion of their significance and implications. Any further discussion of the case must be predicated upon our judgment that the twins are monozygotic. As one with long experience in diagnosing the zygosity of twins, the senior author considers the evidence for the monozygosity so strong that it would be a waste of effort even to summarize or reiterate it. The remainder of this paper will be devoted to a discussion of two main problems that grow out of the facts reported above: I, the significance of the spina bifida condition; and II, the genetic significance of the discordant polydactyly.

I. Significance of the Spina Bifida Syndrome

Medical authorities commonly interpret spina bifida as a classical example of developmental arrest. The idea is that, at a critical moment quite early in embryonic development, during the energetic process of neural tube closure, this process is slowed down and arrested short of completion, leaving the posterior region of the neural groove wide open dorsally. Doubtless the spastic paralysis of the lower limbs is a physiological consequence of the lumbar spina bifida.

When this case was first brought to our attention we were reminded of the report of Keller and Niedoba[3] on double monsters in calf fetuses. In these conjoined twins there was a very high incidence of developmental arrests, especially of spina bifida, hare lip, cleft palate and cryptorchid testes. It was difficult for us to dissociate the cause of these arrests from the cause of twinning itself. If we regard these arrests as due to an early developmental retardation that expresses itself at relatively late periods, it seems probable that it was the same retardation that brought about the twinning itself.

In the majority of cases of human one-egg twinning it would seem that the developmental arrest responsible for twinning has little if any after-effects on the twinned embryos. The two embryonic primordia undergo nearly complete regulation except for vestiges of mirror imaging and each develops into a normal fetus. The earlier the twinning division, the better is the chance for complete regulation and resumption of normal development. When twinning begins relatively late, however, the division process is unable to complete itself and double monsters result, which are characterized by numerous developmental arrests.

In the present case the twins were reported as being enclosed within a common amnion. Amnion formation in man, as in the armadillo and various other mammals is accomplished in an entirely different way from that in birds and in the majority of other mammals, namely, by the hollowing out of a solid ball of ectoderm prior to the establishment of a visible embryonic axis. The hollow in this ectodermic ball constitutes the amniotic cavity. The ectoderm on one side of this hollow ball is thicker than the other and this constitutes the ectodermal part of the embryo, which, though not yet visibly organized into an axis, doubtless is already psysiologically differentiated axially and has the beginnings of a right-left differential. The thinner part of the ectodermic ball becomes the ectodermic component of the amniotic membrane.

When a pair of human twins is found to be enclosed within a common amnion the mode of origin probably resembles that in the armadillo, where it has been shown that if two growth centers arise sufficiently far apart, two separate twins may result; but if they arise too close together, they are likely to remain in contact and to form a double monster or a pair of conjoined twins.

In the present case the twins were completely separate and must therefore have originated from two growth centers far apart in the ectodermic plate. Such a mode of twin origin is probably as late in the embryonic process as it is possible for separate twins to be formed. In fact, it seems probable that our twins barely escaped becoming a double monster, and therefore it is not surprising

that they should, in common with double monsters in general, exhibit pronounced signs of developmental arrest.

The question arises in this case, as in others where twinning and developmental arrests are found to be correlated, whether or not the two phenomena are due to the same causal factor or are independent of each other. Our hypothesis in these cases is that the originally single embryo prior to amnion formation was in some way retarded or brought to a temporary developmental standstill analogous to that normal for the armadillo. When once more the embryo "wakes up" and resumes development, future stages are destined to have been affected by this developmental interruption. Thus, after amnion formation the ectodermic embryonic plate becomes somewhat deaxiated so that two organizing centers instead of the prospective single center arise and two embryos develop. These two embryos apparently show no further signs of retarded development until some time later when the highly energetic process of neural fold closure is interrupted short of completion with the resulting almost identical spina bifida in both twins.

According to this view both spina bifida and twinning are consequences of one and the same developmental retardation. The fact that twinning precedes the expression of spina bifida in no wise precludes the possibility that they may have a common cause, for it is well known that experimental exposures of the eggs of fishes and other forms shortly after fertilization to growth inhibiting agents result much later in twinning and all sorts of developmental arrests including spina bifida. In other words, the effects of early growth retardation do not become evident immediately nor simultaneously, but may appear at various stages of development early or late. In the present case the twinning division was the first effect of an early retardation and a later effect was spina bifida in both twins.

Spina bifida is universally recognized as a developmental arrest, while twinning has been experimentally induced by early temporary slow-downs of development. It therefore seems more probable in the present case that both twinning and spina bifida are consequences of one and the same retarding agent rather than of two such agents operating independently and at different times. Whether the agency in question had a genetic or an environmental basis is a question about which we have no evidence. The chief point we wish to make is that the present case offers one more piece of evidence that one-egg twinning is caused by early temporary growth stoppage or arrested development.

II. Interpretation of Polydactyly in These Twins

Genetic Background of Human Polydactyly. Polydactyly (sometimes called hexadactyly) in man consists of an extra digit on hands or feet. The best known and much the commoner type of polydactyly involves an extra little finger (digit V) or an extra little toe. A far less common type involves an extra thumb or an extra big toe.

The extra-digit-V type of polydactyly is referred to in genetic textbooks the world over as a classic case of a simple dominant trait. In many published pedigrees the trait is shown to be passed directly from affected parent to some of the offspring for several successive generations, but in some instances even in such pedigrees affected offspring are derived from phenotypically normal parents. The trait is so rare that practically all affected individuals are heterozygous for the polydactyl gene. It is not known what a homozygous individual would be like: possibly the homozygote would be lethal. When the trait appears at all, the degree of its expression is highly variable. It may be fully present in both hands and both feet, in hands only, in feet only, in one hand and one foot, or in only one hand or one foot. Also the extra digit may be fully developed or vestigial, being sometimes loosely attached or represented by a fleshy mass of tissue not at all like a finger. Such a character having a genetic basis but varyingly expressed is known technically

as an irregular dominant with fairly high penetrance and highly variable expressivity in the heterozygote.

Various writers, in attempting to account for the above described conditions, have suggested that the variable expression of the trait depends upon individual differences in the genetic milieu or upon modifying effects of external influences during development. Later on we shall show that this explanation fails to account for the appearance of polydactyly in only one of a pair of one-egg twins developing within a common amnion.

The much rarer type of polydactyly, involving an extra thumb or big toe, such as we find in our twin *A*, is not often described in genetic literature. Hefner[2] presented a pedigree of polydactyly associated with extra phalanges in the thumbs. Four members of one generation were affected, but the character had not appeared during three previous generations. Yet Hefner regarded the character as hereditary and probably dominant. There are doubtless other references to extra-thumb polydactyly that have failed to come to our attention.

In his *Principles of Heredity* Snyder[8] in speaking of polydactyly, states that "in the white race this condition is the result of a dominant gene. In Negroes a recessive mutation resulting in a similar condition has occurred." This must be borne in mind in our discussion, for our spina-bifida twins were Negroes.

We have found only three pairs of one-egg twins with polydactyly. Two of these occur in a pedigree published by Odiorne.[7] In both of these pairs the twins had extra digits on both hands and both feet. A third pair of polydactylous one-egg twins consists of a pair of Siamese twin fetuses on exhibition in an anatomical museum. In all these cases an extra fifth digit was involved and it was present on all hands and all feet.

When the physicians' report was first submitted to the senior author no investigation had been made as to the possible heredity of the polydactylous condition in twin *A*. Since it seemed necessary to have this information before attempting to discuss its significance, Dr. Quisenberry undertook to investigate the siblings, parents, grandparents and all other known relatives of the spina-bifida twins and found not a single case of polydactyly. None of the neighbors knew of any such case in the community. This seems to show without question that we are not dealing here with a dominant trait. Snyder's suggestion, however, that polydactyly in Negroes may be recessive might fit this case. We have no evidence either for or against this possibility, but it cannot be ruled out as an alternative explanation.

The Discordant Incidence of Polydactyly in One-Egg Twins

Assuming that polydactyly in these twins may be hereditary, even if recessive, we still have the problem of accounting for its appearance in only one twin and only in her right hand. Students of ordinary dominant polydactyly try to account for its irregular expression by stating that its appearance depends upon the genetic milieu of the individual or upon differences in external conditions during development. In the case of one-egg twins, however, there can be no difference in genetic milieu between the two individuals nor would there be genetic differences between the two sides of a single individual. Also it is difficult to imagine what sort of differences in external conditions could exist for twins developing within a common amnion. If there had existed such differences they should have produced some differences in body weights, in the extent of spina bifida or in tooth eruption. If we must call upon any sort of environmental differences to account for the peculiar distribution of polydactyly in these twins, these differences must be much more subtle than anything contemplated by geneticists hitherto. The environmental differential must be internal and of such a character as to be able to act unilaterally. Such a differential is known to exist in the form of a right-left physiological difference that results in extensive bilateral asymmetry. It is this differential, we believe, that

explains why certain inherited anomalies sometimes appear in only one member of a pair of one-egg twins or only on one side of one twin or of a single individual.

In the present case there is good evidence that the twinning division took place rather late in development as the result of a failure of the prospective right and left halves of the originally single embryo to unite to form a single axis, but after a right-left differential had been established. This differential, though slight, may have been sufficient to favor the expression of polydactyly in one twin and prevent its appearance in the other. Similarly, when the twin showing polydactyly was in the stage of limb bud formation it seems logical to assume that the right-left differential favored the formation of an extra thumb in the right hand and prevented its development in the left hand.

Parenthetically, it seems appropriate to suggest here that the above explanation of the unilateral appearance of polydactyly in one-egg twins applies equally well to unilateral polydactyly in single individuals who are not twins. It may also apply to many other hereditary anomalies that appear sometimes unilaterally and sometimes bilaterally. It may also be suggested that the total failure of expression of polydactyly and other dominant traits in some heterozygotes may be due to internal physiological conditions operating at critical developmental moments rather than to differences in genetic milieu or to differences in external conditions.

Other Possible Interpretations of the Present Case of Polydactyly

Two other possible interpretations of polydactyly in these twins are permissible. First, the character may be a new mutation. Second, it may be a nonhereditary somatic modification, a "phenocopy" (Goldschmidt) of a well-known hereditary character.

The possibility that the trait is due to a new mutation is remote but cannot be absolutely excluded. Polydactyly is relatively rare in our population. According to Danforth,[1] about one person in a thousand shows the trait. He also estimates that a new mutation from normal to polydactyly occurs in about one in 6,000 genes, just about often enough to balance losses of the gene when polydactylous lines run out without offspring. These frequencies apply to little-finger polydactyly and would have to be greatly reduced to fit double-thumb polydactyly, which is very rare. If the latter is recessive, its appearance would be possible only by the chance union of two normal-fingered but heterozygous parents. If in our twins the character is recessive there is no chance for it to appear in the generation in which the hypothetical new mutation occurred unless in some embryonic cell both allelic genes mutated simultaneously, a contingency so remote as to be hardly worth considering as a possibility. It seems then that we may safely rule out the new mutation interpretation of polydactyly in the present case.

There is still one other possible interpretation, namely, that the double-thumb condition was a somatic modification, a phenocopy, and not hereditary at all. There is evidence that complete or partial limb doubling occurs as the result of a lowered developmental rate, and the double-thumb condition may be the result of a half-abortive attempt at limb doubling, the extra thumb being all there was of the extra hand. If then we regard the apparent polydactylous condition as no more than a belated expression of twinning of a limb bud, its cause was more than likely connected with the cause of other conspicuous developmental arrests so strikingly present in these twins. To account for the appearance of the condition in only one twin and in only the right hand of that twin requires the same sort of physiological interpretation as if the character were hereditary, and there is no need to repeat it here. In conclusion, let us say that we have no great preference for either of the two suggested interpretations of the present case of polydactyly. The reader will naturally take his choice or possibly may think of a more satisfactory one. This is a very puzzling case, which is

the main reason for our attempts to elucidate it.

Summary

1. Two physicians report the birth of a pair of Negro twins with one simple placenta and both twins within a common chorion and amnion.

2. The twins were alike in having extensive lumbar spina bifida and spastic parlysis of the legs. They weighed the same and were essentially identical in form and features. They are diagnosed as monozygotic. The only noticeable difference between the twins concerned polydactyly. Twin *A* had an extra thumb on the right hand, twin *B* was normal.

3. It is concluded that spina bifida was due to a developmental slow-down prior to the twinning division and that the same arrest probably caused the twinning.

4. The polydactylous conditions is also discussed from the genetic point of view; the possibilities that it is due to dominant mutation, a recessive gene, or to a belated partial limb doubling, are considered.

Literature Cited

1. DANFORTH, C. H. Chapter *in Eugenics, Genetics and the Family*. Baltimore, 1923.
2. HEFNER, R. A. *Jour. Hered.* 15:323-29. 1924.
3. KELLER, K., and T. NIEDOBA. *Zeit F. Zuchtung*, 37. 1937.
4. NEWMAN, H. H. *The Physiology of Twinning*. Chicago, 1923.
5. ————. *Human Biol.* 12:1. 1940.
6. ————. *Multiple Human Births*. New York, 1940.
7. ODIORNE, J. N. *Jour. Hered.* 34:2. 1943.
8. SNYDER, L. H. *The Principles of Heredity*. New York, 1940.

✦

A STUDY OF RACE

THE significance of genetics for problems of evolution had been recognized very early. Nevertheless there has been for a long time a rather strict division of labor between the experimental geneticist and the student of speciation. Results derived from breeding experiments with a few favored organisms were used in the interpretation of the phenomena which the systematists encountered in wide ranges of other organisms. Rarely was the attempt made to concentrate both genetic and taxonomic efforts on one and the same organism, as for instance in Goldschmidt's pioneer studies of geographic variation of the gypsy moth *Lymantria*.

The study by Dobzhansky and Epling* represents the most thorough contribution, at a new level, to this approach. The genetic analysis of the difference between the geographic races of *Lymantria* had been based on small numbers of individuals of each group. Basically, a single individual was regarded as a representative of each race and emphasis was placed on the genetic differences which distinguish such representatives. Races, however, are not units of individuals which possess identical genotypes. They are groups of individuals of different constitution and are distinguished from another most frequently by characteristically different frequencies of alternative genetic compositions rather than by absolute, all-or-nothing characteristics. Thus the student of population genetics, taking his lead from the concepts derived by Haldane, Fisher, and Wright, is led to studies of large samples of populations from wide geographic ranges.

The morphological and physiological properties which distinguish different geographic races are mostly slight. An orthodox analysis in terms of Mendelian factors is very laborious and indeed would easily become impossible if at-

*DOBZHANSKY, TH., and C. EPLING. Contributions to the Genetics, Taxonomy and Ecology of *Drosophila pseudoobscura* and its relatives. *Carnegie Inst. Washington Publ. 554*, 183 pp. 1944.

tempted on a sufficiently large scale. It can be replaced, however, by a cytologic study of alternative chromosome configurations. Here, the discovery that the sequence of bands in the salivary chromosomes of diptera is not invariant for all individuals of a species but occurs in nature in a number of variations, offers an alternative tool for the study of populations. Chromosomes normally reproduce exact replicas of themselves. They form a continuous series of descent as do genes. If a population consists of individuals of different homo- and heterozygous combinations of alternative chromosomal architectures, an analysis by means of the microscope and a statistical evaluation of the data gained is equivalent to the unattainable gene analysis. Indeed it is superior to it in an important aspect. Identity of alleles may well be due to independent origin by recurrent mutation, not only to common descent. Identity of sequence in chromosomal bands can be shown most likely to be due to common descent only, since the probability of any one sequence arising from another is the product of those of several processes each of which has a very low probability itself.

In addition to some related species, the main organisms with which the authors are concerned were formerly known as *Drosophila pseudoobscura*, races *A* and *B*. They were first distinguished by Lancefield on the basis of high sexual isolation, sterility of male hybrids, and lowered fertility of the offspring from backcrosses of F_1 females. Differences in band sequence of certain chromosomes were for some time the only morphological differences by means of which *A* and *B* could be separated. Recently, however, certain differences in wing measurements and in number of teeth of sexcombs in the male furnish external criteria for taxonomic diagnosis. Since it can be shown by means of a study of chromosome morphology that the two groups in nature, while widely overlapping in distribution, are reproductively isolated from each other, the authors decide to describe the two "races" as species reserving the name *D. pseudoobscura* for the old "race *A*" and giving the new name, *D. persimilis*, to "race *B*". If it seems unusual to describe species on the basis of chromosomal studies, the defense is made that "the methods of systematics in any group are determined by the level which the knowledge of this group has attained and by the uses to which this knowledge is being put." The geographic range of the two species extends from British Columbia to Guatemala (*D. pseudoobscura*) or California (*D. persimilis*) and from the Pacific Coast to the Rocky Mountains and Texas (*D. pseudoobscura*) or the Sierra Nevada-Cascades Mountain chain (*D. persimilis*).

An understanding of the dynamics of populations presupposes a knowledge of ecological factors, habitat, food of larvae and adults, attractive radius of food, migrating habits, seasonal cycles, etc. Laboriously and ingeniously the authors have worked to clarify some of these factors but they would be the last to maintain that complete answers have been obtained on each point.

The first section of the book "Taxonomy, geographic distributions, and ecology of *Drosophila pseudoobscura* and its relatives," by Dobzhansky and Epling covers 46 pages. The second section, "Chromosomal races in *Drosophila pseudoobscura* and *Drosophila persimilis*," by Dobzhansky, comprises nearly 100 pages. It is based on a stupendous amount of labor and a searching analysis of the factual results.

The opportunitity to study chromosomal races in the two species is offered primarily by the great variability in band sequence in the third chromosome. Fifteen different arrangements are known in natural populations of *D. pseudoobscura* and seven in *D. persimilis*. Other chromosomes show similar differences, particularly the X-chromosome whose band arrangements are correlated with the presence or absence of the peculiar sex-ratio phenomenon which results in the production, by males, of nearly exclusively female offspring. Strangely enough, however, the variability of the third chromosome surpasses

VARIATION IN CHLOROPHYLL DEFICIENCY

Figure 10

The lettuce plants on the left and in the center are typical of the type of albinism described under Case 3. Note the irregular pattern of the chlorophyll deficient areas. The normal green plant on the right is the same age as the chlorophyll deficient plants.

ization of the pigment in the cotyledons. The expanded cotyledons have a narrow border of tissue about 1/16-1/8 inch in width around the entire margin which is completely devoid of pigment. These plants are quite conspicious in a segregating progeny when contrasted with the normal yellow-green plants in which the entire cotyledon is ordinarily pigmented. This character is most easily scored at the time the first true leaf is expanding.

The plant described above was crossed with an individual of the Grand Rapids variety. This variety is light green or yellow green as opposed to the dark green types such as the New York and Imperial strains. The particular strain of Grand Rapids used was one given to us by Dr. H. D. Brown of the Ohio Agricultural Experiment Station.

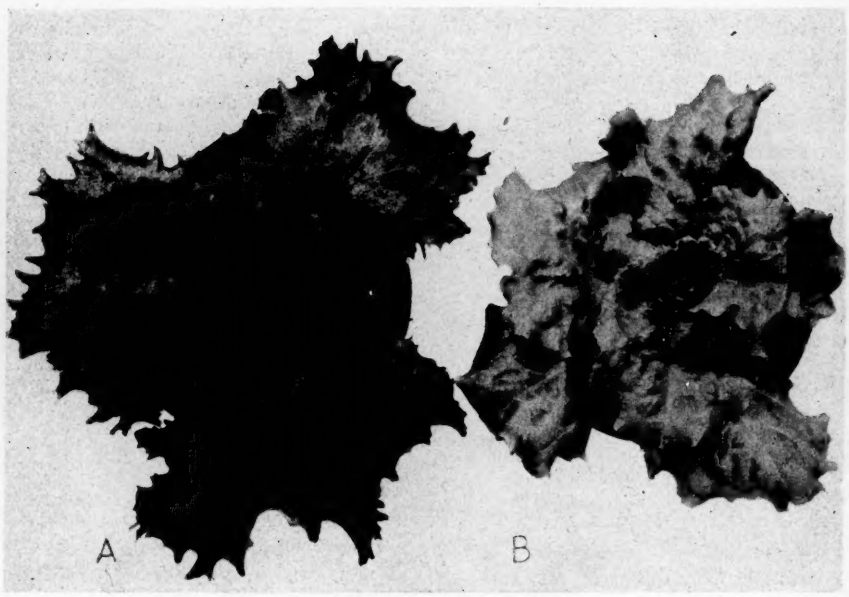
Five F_1 plants matured seed. All were yellow-green with serrate leaf margins characteristic of the Grand Rapids variety. In the five F_2 families, chlorophyll deficiency segregated as indicated in Table I. These data (Table I) indicate that this chlorophyll deficiency appears in a 1:3 ratio and is dependent upon a single gene, recessive to normal yellow-green pigmentation.

Nine F_2 plants were tested in F_3, five with yellow-green pigmentation and four which were chlorophyll-deficient (see Table II). These data verify the conclusion that this deficiency is due to a single recessive gene.

Case 3

In the spring of 1939 a variegated lettuce plant was noticed in a progeny that was being tested for resistance to downy mildew (*Bremia lactucae*). In phenotypic appearance this plant was quite different from the types of albinism described as cases 1 and 2. The leaves were characterized by a blotching or uneven absence of green pigment. Selfed progeny from this plant ranged from normal green individuals to those with barely enough chlorophyll to support life (Figure 10). In most cases plants with pronounced chlorophyll deficiency, were weak and sickly, and died before maturing seed.

This type of albinism is transmitted to the progeny depending upon the degree and intensity of the pigment in the female parent. Normal green individuals selected from the progeny of the original mother plant, when self-pollinated, produce only normal green plants. Medium and highly deficient plants usually produce a varying per-



A CHAMELEON LETTUCE PLANT

Figure 11

The light green chlorophyll deficient plant on the right is described under Case 4. This plant at a later stage of growth became normally pigmented. The plant on the left is a normal green plant from the same progeny as the chlorophyll deficient plant.

centage of variegated plants. The number of variegated plants produced depends upon the degree of albinism of the variegated parent.

The original variegated plant (white-seeded) ♀ was crossed with Imperial 847 (black-seeded) ♂, and five F_1 plants were obtained, two of which produced seed. These two plants had black seed and were therefore hybrids. They were normal green in appearance. The selfed progeny of each of these individuals was also normal green in appearance. These tests indicate that the chlorophyll deficiency of Case 3 is cytoplasmic and transmitted only by the maternal parent.

Case 4

In the spring of 1942 a chlorophyll-deficient plant was discovered in a seed bed of the Imperial 850 strain (Figure 11). This plant was saved, transplanted to a six-inch pot, and transferred to the greenhouse. At about the time it should have developed a head, the plant became strongly pigmented and fully normal in color. It was possible to place the selfed progeny from this plant in three groups according to their degree of chlorophyll deficiency, as follows: (1) dark green plants, (2) light green plants, (3) chlorophyll deficient in the first true leaf. After a month had elapsed the plants were scored again, with the result that those of the third class now passed as normal, the plants of class 2 retaining their light green color. Appropriate tests using a black-seeded plant as a tester indicates that this type of chlorophyll deficiency is cytoplasmic and cannot be transferred through the pollen or egg cells.

Discussion

The variegated deficiency (Case 3) seems to be identical with that de-

scribed by Thompson.[2] The peculiar pattern of the chlorophyll areas and their non-mendelian behavior suggest a similar genetic basis.

Gustafsson[1] has constructed an elaborate system for classifying the chlorophyll mutations of barley and has suggested that it may be usefully applied to chlorophyll mutations in other plants. This system is based upon the pigment pattern, its intensity and genetic nature. If this system is used to classify the four cases of chlorophyll deficiency reported above, Case 1 is typical *albina* (no carotinoids or chlorophyll formed), Case 2 is *alboxantha* (tips white, base yellow) and Cases 3 and 4 are plasm mutations.

It seems fair to conclude from the evidence presented that the chlorophyll inheritance apparatus in lettuce is subject to periodic breakdowns which interfere with the normal functioning of the plant. Of most frequent occurrence are cytoplasmic deficiencies; next are single-gene mutations. It is unlikely that either class of deficiency will become a factor in commercial lettuce production because chlorotic or albino plants are quite conspicious and as a rule are eliminated by roguing in commercial seed fields.

TABLE I.—F_2 segregation from the cross yellow-green × chlorophyll-deficient.

Family No.	Chlor.-def.	Yellow-green	Total	χ^2
15487	156	482	638	0.10
15395	40	92	132	1.88
15488	12	28	40	0.30
15396	96	256	352	0.97
15397	47	158	205	0.47
Total	351	1016	1367	3.72

(df = 5, P = 0.6)

TABLE II.—F_3 segregation from the cross yellow-green × chlorophyll-deficient.

Planting Number	Parent Phenotype	Chlor.-Def.	Yellow-Green	Total	χ^2
15395-1	yellow-green	54	195	249	1.45
15395-2	" "	24	73	97	0.03
15395-3	" "		26	26	
15395-4	chlor.-def.	35	—	35	
30064	" "	282	—	282	
30065	" "	58	—	58	
30061	yellow-green	5	9	14	0.85
30062	" "	7	18	25	0.28
30066	chlor.-def.	8	—	8	

Summary

The phenotypic appearance and genetic behavior of four chlorophyll deficiencies in lettuce are described. Three cases proved to be cytoplasmic and transmitted by the maternal parent and the other was apparently dependent upon the result of a mutation by a single recessive gene. Using the classification of Gustafsson, one case was albina, one was alboxantha, and two were plasma mutations.

Literature Cited

1. Gustafsson, A. *Lunds Univ. N. F. Avd. 2* Bd. 36. No. 11, pp. 1-40. 1940.
2. Thompson, R. C. *U.S.D.A. Tech. Bul. 620.* 1938.
3. ———— *Jour. Agr. Res.* 66:441-446. 1943.

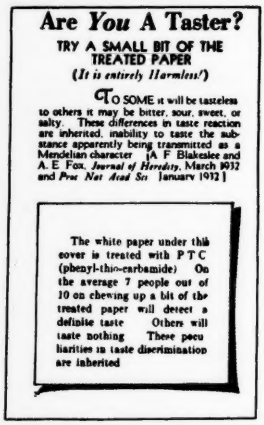
◆

Inbred Sunflowers

Although the data presented show that inbreeding brings about a lowering of yield and vigour in sunflowers, inbreeding is of value in eliminating undesirable genes and in bringing about uniformity for desirable characters such as non-branching strong stems, earliness, small seed size, and high oil content. . . . Morozov and Ananeva of the Saratov Experimental Station report that during 19 years of inbreeding sunflowers no strains materially superior to the original forms had been obtained by selective inbreeding alone. As in the case of corn, the value of inbreeding sunflowers appears to be for production of lines breeding true for desirable characters which can then be used in combinations to produce single cross hybrids.

The high yield of single crosses is contingent upon securing a high degree of crossing between the paired inbred lines. Unless the paired lines differ in pollen yield and, or, self-fertility it appears that both inbred lines will cross to about the same extent and therefore seed from both could be used commercially.

As far as possible it would be desirable to have one or both lines carry marker genes so that the extent of crossing could be determined.—Unrau and White. *Sci. Agr.*, July, 1944.


Are You A Taster?
TRY A SMALL BIT OF THE TREATED PAPER
(It is entirely Harmless!)
TO SOME it will be tasteless to others it may be bitter, sour, sweet, or salty. These differences in taste reaction are inherited, inability to taste the substance apparently being transmitted as a Mendelian character [A F Blakeslee and A. E Fox, Journal of Heredity, March 1932 and Proc Nat Acad Sci January 1932]
The white paper under this cover is treated with P T C (phenyl-thio-carbamide) On the average 7 people out of 10 on chewing up a bit of the treated paper will detect a definite taste Others will taste nothing These peculiarities in taste discrimination are inherited

LEAFLETS TO DEMONSTRATE HEREDITY

Last year the "Taster test" served as an introduction to human genetic differences in many classrooms. The test is so easily made with the treated paper, and arouses such great interest that it appears to have a definite place in group demonstrations of such differences. These leaflets, size 3⅜" by 5½", are available in quantities for those who wish to use them for classroom or lecture purposes. On the back is printed an outline 4-generation pedigree chart. Sample on request. Prices: 10 for 25c; 50 for $1.00.

AMERICAN GENETIC ASSOCIATION

1108 - 16TH STREET, N. W. WASHINGTON 6, D. C.

FEEDS for Your "Lab" Animals

DEPENDABLE feeds are important in the laboratory where tests of various kinds are being made on small animals.

Dickinson, producer of the well known Globe Feeds, is one of the oldest and most reliable feed houses from which to buy your "Lab" animal feed.

Globe Feeds supply complete rations. They are dry . . . easily stored . . . take up little space . . . and involve little waste. They hold the same high standard of quality from year to year.

GENEROUS SAMPLES FREE

To supervisors of experimental animals, we will send a free sample, large enough to make an adequate test. Just tell us the kind and quantity of food desired.

FOR RABBITS

Globe Rabbit Glob-ets to be supplemented with hay.
Globe Rabbit with Alfalfa, meal form—complete.
Globe Rabbit with Alfalfa, pellet form—complete.

FOR RATS AND MICE

Dickinson's Dog Food—Nugget—Pellet or meal form. Dickinson's Kibbled Dog Food.

FOR GUINEA PIGS

Globe Rabbit Glob-ets to be supplemented with hay.
Globe Rabbit with Alfalfa, meal form—complete.
Globe Rabbit with Alfalfa, pellet form—complete.
Supplement with Vitamin C carrier three times a week.

THE ALBERT DICKINSON COMPANY

2714 WEST 35TH STREET CHICAGO 90, ILLINOIS

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks.

Its membership is composed of men of science, teachers, publicists, physicians, clergymen, parents, students, horticulturists, and breeders of live stock throughout the world.

The Association owns the JOURNAL OF HEREDITY, which is published monthly and sent to each member without additional cost. Every member is thus a part owner of the JOURNAL.

Membership imposes no burdensome obligations. The Association, which is co-operative in nature, welcomes assistance in research, but does not demand it. Members are invited to submit discussions of the results of their research, accompanied by new and unusual photographs. All papers received will be given full consideration by the editorial board.

Manuscripts should be sent to the Editorial Office of the JOURNAL, Room 205, 1108 Sixteenth Street, N. W., Washington 6, D. C.

Proof: In order to facilitate prompt publication only galley proof will be sent to authors.

Reprints: On request the author will receive gratis 100 reprints without covers. Covers and additional copies will be furnished at cost.

REQUIREMENTS FOR MEMBERSHIP

Subject to the approval of the Council, any person interested in the improvment of the human race or the creation of better varieties of plants and animals, is eligible for membership.

The Association welcomes all who are interested in its program, and the Secretary will be glad to answer any inquiries.

Annual dues, giving the right to attend all meetings, and receive the JOURNAL OF HEREDITY, are $3 within the United States and its possessions; $3.25 in Canada, and $3.50 in all other foreign countries; life membership, $50. Subscription to the JOURNAL is $3.50 per year (foreign postage extra).

If you are not already a member, and want to become one, or if you know of anyone who would be interested in membership, write to

THE AMERICAN GENETIC ASSOCIATION

Washington 6, D. C.